UNITED STATES DISTRICT COURT SOUTHERN DISTRICT OF NEW YORK

Michelle Mascioli
Plaintiff(s),   **Case #**

v.

Chevron U.S.A., Inc., Liberty Utilities (New York Water) Corp.,
Defendant.

COMPLAINT

Jury Trial Demanded

Introduction

1. This is a toxic tort action brought by Plaintiff against Chevron U.S.A. Inc., formerly known as Gulf Oil, and Liberty (American) Water for injuries and damages resulting from prolonged exposure to hazardous chemicals, including Trichloroethylene (TCE), Vinyl Chloride, and Methyl tert-butyl ether (MTBE). These contaminants were introduced into the environment due to the negligent actions of Chevron, which caused an oil spill at a terminal in Oceanside, New York, and were subsequently exacerbated by Liberty Water's failure to provide adequate water quality monitoring, reporting, and timely public notification.

2. A Record of Decision dated Decemeber 2021 presents the remedy for the Former Gulf Oil Terminal site, a Class 2 inactivehazardous waste disposal site. The remedial program was chosen in accordance with the New York State Environmental Conservation Law and Title 6 of the Official Compilation of Codes, Rules and Regulations of the State of New York (6 NYCRR) Part 375, and is not inconsistent with the National Oil and Hazardous Substances Pollution Contingency Plan of March 8, 1990 (40CFR300), as amended.
    This decision is based on the Administrative Record of the New York State Department of Environmental Conservation (the Department) for the Former Gulf Oil Terminal site and the public's input to the proposed remedy presented by the Department.

3. The public input period for information and questions related to this site was held during Covid lockdown. There was insufficient notice to the public and the time period chosen was done so to avoid or circumvent the necessary requirement of a duty to inform the public. NYSDEC Announced an "Extension of Public Comment Period for Proposed Remedial Action Plan for the Former Gulf Oil Terminal State Superfund Site" .

The initial public comment period for the work plan was to end on **March 29, 2021. And then e**xtended. To **April 28, 2021**. ("Former Gulf Oil Terminal Site (Oceanside) - Public Comment EXTENDED for Proposed Site Cleanup", NYSDEC, March 31, 2021. https://content.govdelivery.com/accounts/NYSDEC/bulletins/2ca954d)

Jurisdiction and Venue

 2. This Court has jurisdiction over this action pursuant to 28 U.S.C. §1331 (federal question jurisdiction) and 28 U.S.C. §1367 (supplemental jurisdiction).

 3. Venue is proper in this Court pursuant to 28 U.S.C. §1391 because the events giving rise to this Complaint occurred within the Southern District of New York.

214-F - Action to Recover Damages for Personal Injury Caused by Contact With or Exposure to Any Substance or Combination of Substances Found Within an Area Designated as a Superfund Site.

Universal Citation:

NY CPLR § 214-F (2023)

§ 214-f. Action to recover damages for personal injury caused by contact with or exposure to any substance or combination of substances found within an area designated as a superfund site. Notwithstanding any provision of law to the contrary, an action to recover personal damages for injury caused by contact with or exposure to any substance or combination of substances contained within an area designated as a superfund site pursuant to either Chapter 103 of Section 42 of the United States Code and/or section 27-1303 of the environmental conservation law, may be commenced by the plaintiff within the period allowed pursuant to section two hundred fourteen-c of this article or within three years of such designation of such an area as a superfund site, whichever is latest.

Parties

 4. Plaintiff [Name(s)], a resident of Oceanside, New York, has suffered significant health problems, including thyroid disorder, ulcerative colitis, heart and cardiac damage, kidney disease, and cancer, as a result of exposure to TCE, Vinyl Chloride, and MTBE. 5. Defendant Chevron U.S.A. Inc., formerly known as Gulf Oil, is a corporation engaged in oil and gas operations and has its principal place of business at [Insert Address]. 6. Defendant Liberty (American) Water is a public utility responsible for providing water services to Oceanside residents.Potentially Responsible Parties (PRPs) are those who may be legally liable for contamination at a site. The PRPs for the site, documented to date, include:

Chevron U.S.A., Inc. (formerly Gulf Oil)

A Brownfield Cleanup Agreement (C130165) was signed by the Department with Lowe's HomeCenters, Inc. as the volunteer on May 11, 2007. Subsequently, due to disagreements betweenLowe's Home Centers, Inc. and Chevron U.S.A., Inc., the Brownfield Cleanup Agreement was

terminated on June 13, 2008. The Department subsequently listed the site as Class 2 on the NYSRegistry of Inactive Hazardous Waste Disposal Sites on September 8, 2008.

The Department and Chevron U.S.A., Inc., entered into a Consent Order on December 23, 2009. The Order obligates the responsible party to implement a full remedial program.

Factual Background

7. In or about [specific date or year], an oil spill occurred at a Chevron-operated terminal in Oceanside, New York, releasing hazardous chemicals into the surrounding environment. 8. The spilled substances included TCE, Vinyl Chloride, and MTBE, which are known to pose significant risks to human health, including carcinogenicity, endocrine disruption, and organ toxicity. 9. The contaminants infiltrated local water supplies and soil, thereby exposing residents to prolonged chemical exposure. 10. Plaintiff and other Oceanside residents consumed and were exposed to contaminated water and air over an extended period, unknowingly placing their health at severe risk. 11. Liberty (American) Water failed to adequately test for these contaminants and did not provide accurate or timely water quality reports to residents. 12. Public notification of potential contamination was insufficient and occurred during the COVID-19 lockdown, further suppressing critical information and preventing residents from taking protective measures. 13. Liberty Water's failure to warn and inform residents of contamination constitutes gross negligence and a breach of its duty to provide safe and clean drinking water.

SITE DESCRIPTION AND HISTORY

Location: The 7.2-acre Former Chevron/Gulf Petroleum Terminal property is located at 1Industrial Place, Oceanside, Town of Hempstead, Nassau County. The site is bordered to the west by Long Island Railroad tracks, to the north by a former petroleum terminal, to the west byHampton Road and Industrial Place and to the south by a surface water body called Barnum'sChannel.

Site Features: The site is relatively flat and is comprised mainly of paved parking for a wholesalewarehouse facility, approximately 20% of which is on-site. In addition, an operating gas station islocated on the southern portion of the site. Limited landscaping exists mainly along the westernside of the site.

Current Zoning/Use: The site is currently active and is zoned for commercial use. The surrounding parcels are currently used for a combination of commercial and industrial. The nearest residential area is approximately 0.25 miles to the northeast across Daly Boulevard.

Past Uses of the Site: The Former Chevron/Gulf site operated from 1932 until the 1990s as a

petroleum storage terminal. The site previously held nine large-quantity aboveground storage

tanks (ASTs) containing fuel oil, kerosene and gasoline; two small 550-gallon ASTs containing

fuel oil for the on-site garage and office building; three underground storage tanks (USTs)

containing fuel oil (one 550-gallon, one 1,000-gallon and one 5,000-gallon); one 1,000-gallon UST

containing waste oil; a loading rack; a retention pond; a maintenance garage; and an office

complex. Four of the nine large ASTs were demolished prior to 2000, with the remaining five large

ASTs reportedly demolished in 2003. The two 550-gallon ASTs containing fuel oil for the

maintenance garage and the office building were demolished in 2005.

Site Geology and Hydrogeology: Subsurface soil conditions encountered during previous

environmental and geotechnical investigations determined the site lithology to consist of the

following: sand fill from the ground surface to approximately eight feet below ground surface

(bgs); meadow mat (silt with fibrous organics and trace clay) to approximately 15 feet bgs.
Record of Decision, (Former Gulf Oil Terminal, State Superfund Project, Oceanside, Nassau
County, Site No. 130165, New York State Department of Environmental Conservation, 41
pages, December 2021.

https://extapps.dec.ny.gov/data/DecDocs/130165/ROD.HW.130165.2021-12-10.ROD%20Form
er%20Gulf%20Oil%20Site.pdf)

Contaminants
14. Exposure to TCE has been linked to thyroid disorders, kidney damage, and cancer. 15.
Vinyl Chloride is a potent carcinogen associated with liver cancer and other malignancies. 16.
MTBE exposure has been shown to cause neurological symptoms, kidney disease, and
potential cardiac issues. 17. Plaintiff and other residents have developed serious medical
conditions consistent with exposure to these chemicals, including but not limited to thyroid
disorder, ulcerative colitis, heart and cardiac damage, kidney disease, and cancer.

Summary of the Remedial Investigation

A Remedial Investigation (RI) has been conducted. The purpose of the RI was to define the nature

and extent of any contamination resulting from previous activities at the site. The field activities

and findings of the investigation are described in the RI Report.

The following general activities are conducted during an RI:

• Research of historical information;

• Geophysical survey to determine the lateral extent of wastes;

• Test pits, soil borings, and monitoring well installations;

• Sampling of waste, surface and subsurface soils, groundwater, and soil vapor;

• Sampling of surface water and sediment; and

• Ecological and Human Health Exposure Assessments.

The analytical data collected on this site includes data for:

- groundwater

- soil

- soil vapor

RI Results

The data have identified contaminants of concern. A "contaminant of concern" is a hazardous

waste that is sufficiently present in frequency and concentration in the environment to require

evaluation for remedial action. Not all contaminants identified on the property are contaminants

of concern. The nature and extent of contamination and environmental media requiring action are

summarized in Exhibit A. Additionally, the RI Report contains a full discussion of the data. The

contaminants of concern identified at this site are:

methylene chloride

trichloroethene (TCE)

1,2,4-trimethylbenzene

toluene

benzene

ethylbenzene

n-propylbenzene

xylene (mixed)

naphthalene

butylbenzene

methyl-tert-butyl ether (MTBE)

mercury

arsenic

cis-1,2.dichloroethene (cis-DCE)

trans-1,2-dichloroethene (trans-DCE)

phenol

tetrachloroethene (PCE)

vinyl chloride

Summary of Environmental Assessment

This section summarizes the assessment of existing and potential future environmental impacts

presented by the site. Environmental impacts may include existing and potential future exposure

pathways to fish and wildlife receptors, wetlands, groundwater resources, and surface water.

Based upon the resources and pathways identified and the toxicity of the contaminants of

ecological concern at this site, a Fish and Wildlife Resources Impact Analysis (FWRIA) was

deemed not necessary for the site.

Nature and Extent of Contamination:

The sources of the site contamination are associated with historical petroleum terminal operations

conducted from 1931 to the early 1990s, and discharges from piping associated with former ASTs,

CAUSATION:

VINYL CHLORIDE

There is *sufficient evidence* in humans for the carcinogenicity of vinyl chloride. Vinyl chloride causes angiosarcoma of the liver, and hepatocellular carcinoma.

There is *sufficient evidence* in experimental animals for the carcinogenicity of vinyl chloride.

There is *sufficient evidence* in experimental animals for the carcinogenicity of chloroethylene oxide.

There is strong evidence that the carcinogenicity of vinyl chloride operates by a genotoxic mechanism that involves metabolic activation to reactive metabolites, binding of the metabolites to DNA, promutagenic action of these adducts leading to mutations in proto-oncogenes and tumour-suppressor genes. Many of these key events identified in experimental animals have also been demonstrated in humans.

Vinyl chloride is *carcinogenic to humans (Group 1)*.

Numerous studies on the toxicokinetics, metabolism, genotoxicity, and molecular biology of vinyl chloride provide strong evidence that the carcinogenicity of this chamical involves a genotoxic mechanism of action, mediated by reactive metabolites. The extensive information on the mechanism underlying vinyl chloride-induced carcinogenicity has established many key events in the pathway of vinyl chloride-induced liver carcinogenesis. These key events include metabolic activation to reactive metabolites, binding of the metabolites to DNA, promutagenic action of these adducts leading to $G \rightarrow A$ and $A \rightarrow T$ transitions, and the effects of such mutations on the functioning of proto-oncogenes and tumour-suppressor genes at the gene and protein levels, with tumourigenesis as the final outcome. Many of these key events identified in experimental animals have also been demonstrated in humans.The carcinogenicity of vinyl chloride has been studied intensively and repeatedly in experimental animals, with a wide range of concentrations, spanning orders of magnitude. The many studies consistently showed hepatic and extrahepatic angiosarcomas in mice and rats. Various other malignant neoplasms also occurred at several anatomical sites.

Vinyl chloride was evaluated in previous IARC Monographs (IARC, 1979, 1987, 2008) and was classified in Group 1 based on increased risks for ASL and hepatocellular carcinoma (HCC).

A report of three cases of ASL in men who had been employed in the manufacture of PVC resins provided the first evidence of an association between vinyl chloride and cancer in humans (Creech & Johnson, 1974). The case report was particularly informative because of the extreme rarity of this tumour in the general population. The Working Group in 1974 already considered this observation to provide evidence of a causal relationship.

Epidemiological evidence for the carcinogenicity of vinyl chloride in humans derives principally from two large, multicentre cohort studies, one of which was carried out in the USA and the other in Europe. These investigations focused on plants that manufactured vinyl chloride monomer, polyvinyl chloride or polyvinyl chloride products. In addition to reports that pertained to

these cohorts in their entirety, several studies reported findings from individual subcohorts. Results for subcohorts are given in the Tables, but only when they provide important information that is not available in analyses of the full cohorts. Results on six cohort studies have also been reported, in addition to and separate from the two multicentre investigations.

The first published report of the North-American multicentre cohort study (Cooper, 1981) included 10 173 workers from 37 plants. Among the 37 plants included in the study, 11 plants with 1214 workers produced only VCM, 18 plants with 6848 workers produced only PVC, three plants with 935 workers produced both VCM and PVC and five plants with 1176 workers produced homopolymers and copolymers. To be eligible for inclusion into the cohort, male employees at the 37 participating plants were required to have been exposed to VCM for at least one year before 31 December 1972 and to have been employed in or after 1942. A second major update of this cohort was published by Wong et al. (1991). A third major follow-up included 10 109 subjects and provided an update of the vital status through to 31 December 1995 (Mundt et al., 2000).

The European cohort study was conducted in four countries (Italy, Norway, Sweden and the United Kingdom). It included workers from 19 factories: 11 of these produced VCM/PVC, two produced VCM only, five produced PVC only and one was a PVC-processing plant. Male workers who had been employed for at least one year in 1942–1972 in jobs that entailed exposure to VCM were included (Simonato et al., 1991). An update of the study (Ward et al., 2001) analysed incidence and mortality through to the latest year for which data were available in each country, which ranged between 1993 and 1997.

There is compelling evidence that exposure to vinyl chloride is associated with angiosarcoma of the liver, and strong evidence that it is associated with hepatocellular carcinoma. Together with the observation that vinyl chloride increases the risk for liver cirrhosis, which is a known risk factor for hepatocellular carcinoma, the findings from two large multicentre cohort studies provide convincing evidence that vinyl chloride causes hepatocellular carcinoma as well as angiosarcoma of the liver. There is contradictory evidence that exposure to vinyl chloride is associated with malignant neoplasms of connective and soft tissue, and inconsistent or scanty evidence that it is associated with cancers of the lung, brain, lymphohaematopoietic system, and breast, or with melanoma of the skin.

MTBE

Information on the noncancer toxicity of MTBE comes primarily from studies in laboratory animals, however, a few controlled exposure studies, epidemiological studies of humans exposed to gasoline containing MTBE, and side effects reported in patients given MTBE via a tube inserted into their gallbladder for gallstone dissolution contribute to the identification of primary toxicity targets. Therewere 88 laboratory animal toxicity studies with health effects data identified: 42 inhalation, 40 oral, and6 dermal.

The most sensitive noncancer effects in laboratory animals following inhalation exposure appear to be respiratory, neurological, and hepatic effects. Other noncancer toxicityeffects are generally only observed at or above concentrations associated with overt signs of clinicaltoxicity (central nervous system [CNS] depression), including decreased body weight, and endocrine(adrenal), renal, immunological, female reproductive, and developmental effects. Ocular irritation wasalso reported in several inhalation studies; however, this effect is attributed to direct contact with vapors as opposed to systemic effects attributable to inhalation exposure. The mostsensitive noncancer effects in laboratory animals following oral exposure include hepatic, neurological,lymphoreticular, and male reproductive effects. As with inhalation exposure, other noncancer toxicity effects are generally only observed at or above oral doses associated with overt signs of clinical toxicity

(CNS depression), including decreased body weight, and endocrine (adrenal), respiratory, hematological,and renal effects. Gastrointestinal effects consistent with irritation of the gastric mucosa were also observed in gavage studies; however, these findings may not be relevant endpoints for environmentalexposures, in which oral exposure is expected to be predominantly via drinking water.

Respiratory Effects. Some occupational and population-based studies conducted in the early 1990s observed respiratory symptoms with introduction of MTBE into fuel during the oxyfuel program (Alaska DHSS 1992a, 1992b; Moolenaar et al. 1994; Wisconsin DHSS 1995), while other studies did not observe such symptoms (CDC 1993a, 1993b; Gordian et al. 1995; Mohr et al. 1994). However, no clear conclusions can be drawn from these studies due to several limitations. Il Very high inhalation levels associated with lethality resulted in hyperpnea, labored breathing, and respiratory failure (ARCO 1980; Bevan et al. 1997a).

Gastrointestinal Effects. Numerous human studies in patients receiving intracystic MTBE therapy for gallstone dissolution report gastrointestinal side effects, including vomiting, nausea, anorexia, emesis,duodenitis, retching, upper abdominal burning sensation during infusion, gas, and duodenal ulcer (see Section 2.6 for citations). Several epidemiology studies also report nausea and/or vomiting with

inhalation exposure to gasoline containing MTBE; however, these symptoms are likely related toneurological effects associated with MTBE exposure (see Neurological Effects below). In animals, the gastrointestinal tract appears to be a target of toxicity following exposure to high gavage doses, including diarrhea and inflammation of the gastrointestinal tract (Amoco 1992; Robinson et al. 1990);gastrointestinal effects were not observed in animals in drinking water or inhalation exposure studies.

Observed effects in humans and animals are consistent with irritative effects on the gastrointestinal mucosa. Effects associated with intracystic MTBE therapy or bolus gavage exposure in animals may not be relevant endpoints for environmental exposures, in which oral exposure is expected to be predominantly via drinking water.

Hepatic Effects. Numerous human studies in patients receiving intracystic MTBE therapy for gallstone dissolution report hepatic side effects in cases of accidental overflow of MTBE or bile leakage during the procedure, including slight elevations of serum aminotransaminases, increased bilirubin, and alterations in bile duct structure or function (see Section 2.9 for citations). In animal studies, elevated liver weight, hepatocellular hypertrophy, and induction of hepatic enzymes were consistently observed at high exposure levels associated with overt clinical signs of toxicity (e.g., CNS depression) following inhalation (Bevan et al. 1997a; Bevan et al. 1997b; Bird et al. 1997; Dodd and Kintigh 1989; Lington et al. 1997; Moser et al. 1996; Texaco Inc. 1981) or oral (Amoco 1992; Dong-mei et al. 2009; de Peyster et al. 2003, 2014; Robinson et al. 1990; Williams et al. 2000) exposure. These effect may represent adaptive changes following MTBE exposure and are of uncertain toxicological significance. Elevated serum cholesterol was also observed in some oral studies (Robinson et al. 1990; Saeedi et al. 2017); however, the biological significance of this is also unclear due to lack of associated hepatic lesions (e.g., fattyliver).

Renal Effects. One case report indicates renal side effects in a patient receiving intracystic MTBE therapy for gallstone dissolution following accidental overflow of MTBE during the procedure (Ponchon et al. 1988); no renal side effects were noted in other case reports (Allen et al. 1985a; Uchida et al. 1994).

No additional human data are available. Renal toxicity has been consistently observed in male rats at exposure levels at or below those associated with overt clinical signs (e.g., CNS depression) following inhalation (Bird et al. 1997; Lington et al. 1997; Prescott-Mathews et al. 1997) and oral exposure (Amoco

1992; Bermudez et al. 2012; Dodd et al. 2013; Robinson et al. 1990; Williams et al. 2000). Findings in male rats are likely due, in part, to α2u-globulin accumulation, which is not relevant to human health

(Ahmed 2001; Bogen and Heilman 2015; McGregor 2006; Phillips et al. 2008). Renal toxicity (elevated kidney weights, increased incidence and severity of chronic progressive nephropathy) has also been reported in female rats via an unknown mechanism(s); however, findings were less severe and/or at

higher exposure levels compared to male rats (exposure levels at or above those associated with overt clinical signs of toxicity) (Bird et al. 1997; Dodd et al. 2013).

Lymphoreticular Effects. No human data are available. Data from inhalation and oral studies inlaboratory animals provide limited evidence of proliferation of lymphoreticular tissues in rats (Belpoggi et al. 1995, 1997; Lington et al. 1997). These lesions may be preneoplastic in nature (see Cancer Effects below).

Neurological Effects. Some occupational and population-based studies conducted in the early 1990 observed effects consistent with transient CNS depression with introduction of MTBE into

fuel during the oxyfuel program, including headache, nausea or vomiting, dizziness, and a feeling of spaciness or disorientation (Alaska DHSS 1992a, 1992b; CDC 1993a; Moolenaar et al. 1994; Wisconsin DHSS 1995), while other studies did not observe such effects (CDC 1993b; Gordian et al. 1995; Mohr et al. 1994).

Effects consistent with CNS depression have also been reported in patients following intracystic MTBE therapy for gallstone dissolution (see Section 2.15 for citations). No subjective symptoms or alterations in neurobehavioral tests were observed in volunteers following acute-duration exposure to low air levels Renal Effects. One case report indicates renal side effects in a patient receiving intracystic MTBE therapy for gallstone dissolution following accidental overflow of MTBE during the procedure (Ponchon et al. 1988); no renal side effects were noted in other case reports (Allen et al. 1985a; Uchida et al. 1994).

No additional human data are available. Renal toxicity has been consistently observed in male rats at exposure levels at or below those associated with overt clinical signs (e.g., CNS depression) following inhalation (Bird et al. 1997; Lington et al. 1997; Prescott-Mathews et al. 1997) and oral exposure (Amoco 1992; Bermudez et al. 2012; Dodd et al. 2013; Robinson et al. 1990; Williams et al. 2000). Findings in male rats are likely due, in part, to α2u-globulin accumulation, which is not relevant to human health (Ahmed 2001; Bogen and Heilman 2015; McGregor 2006; Phillips et al. 2008). Renal toxicity (elevatedkidney weights, increased incidence and severity of chronic progressive nephropathy) has also beenreported in female rats via an unknown mechanism(s); however, findings were less severe and/or at higher exposure levels compared to male rats (exposure levels at or above those associated with overt clinical signs of toxicity) (Bird et al. 1997; Dodd et al. 2013). Renal effects included in

Claims for Relief
Count I: Negligence (Chevron U.S.A. Inc.)
18. Defendant Chevron's negligence in preventing and responding to the oil spill directly caused the release of hazardous chemicals into the environment, resulting in Plaintiff's injuries.

Count II: Negligence (Liberty Water)
19. Defendant Liberty Water's failure to adequately monitor, report, and notify residents of water contamination constitutes gross negligence, exacerbating the harm caused by Chevron's oil spill.

Count III: Failure to Warn
20. Both Defendants failed to provide sufficient warnings and public notifications, depriving residents of the opportunity to mitigate harm.

Count IV: Breach of Duty
21. Liberty Water breached its duty to provide safe drinking water and to adequately inform residents of any potential risks.

Prayer for Relief

WHEREFORE, Plaintiff respectfully requests that this Court enter judgment against Defendants as follows:

- Compensatory damages for Plaintiff's medical expenses, pain, and suffering.
- Punitive damages to deter similar conduct in the future.
- Injunctive relief requiring Liberty Water to implement comprehensive water quality testing and reporting.
- An extension of time for discovery and to find appropriate attorney representation.
- Any other relief deemed appropriate by this Court.

Dated: December 31, 2024

Respectfully Submitted,

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Link to all documents related to site:

https://extapps.dec.ny.gov/data/DecDocs/130165/

Record of Decision, Former Gulf Oil Terminal, State Superfund Project, Oceanside, Nassau County, Site No. 130165, New York State Department of Environmental Conservation, 41 pages, December 2021.

https://extapps.dec.ny.gov/data/DecDocs/130165/ROD.HW.130165.2021-12-10.ROD%20Former%20Gulf%20Oil%20Site.pdf

"Chevron Environmental Management Company, Remedial Investigation Data Summary Report, 1457 pages, May 2011.
https://extapps.dec.ny.gov/data/DecDocs/130165/Report.HW.130165.2011-05-01.Remedial_Investigation_Data_Summary_Report.pdf

Causation: Toxicology Reports for Major Contaminants

<u>MTBE METHYL tert-BUTYL ETHER</u>

https://wwwn.cdc.gov/TSP/ToxProfiles/ToxProfiles.aspx?id=228&tid=41

"Toxicological Profile for Methyl tert-Butyl Ether (MTBE)", ATSDR. 2023.
https://www.atsdr.cdc.gov/toxprofiles/tp91-c1.pdf

https://www.ncbi.nlm.nih.gov/books/NBK601216/

<u>VINYL CHLORIDE</u>

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https://www.ncbi.nlm.nih.gov/books/NBK304420/

"TOXICOLOGICAL REVIEW OF VINYL CHLORIDE" , Environmental Protection Agency, May 2000.
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"Health effects linked with trichloroethylene (TCE), tetrachloroethylene (PCE), benzene, and vinyl chloride exposure", ATSDR, November 12, 2024
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Bolt HM, Filser JG, Laib RJ, Ottenwälder H. Binding kinetics of vinyl chloride and vinyl bromide at very low doses. Arch Toxicol Suppl. 1980;3:129–142. [PubMed]

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